limitation of experimental models. We do agree that extrapolation to humans has to be done cautiously, as discussed in our study.

The main point of our study was to demonstrate that electrical capture of myocardium, under specific circumstances, can occur after neuromuscular incapacitating device (NID) discharge. Unless the recording system is shielded from electromechanical interference, accurate assessment of cardiac effects of discharges is not possible, and conclusions about the safety of discharges are unreliable. We did not draw conclusions about reports of the possible consequences of NID discharges reported in the media, but we wanted to highlight the potential for myocardial capture and potential risks of high-frequency cardiac stimulation under specific (and likely very uncommon in usual use) circumstances. We agree that it is not possible to directly extrapolate our results to NID use in humans.

The letter by Kroll et al. highlights the same difficulties, with regard to coming to safety conclusions based on the absence of objectively documented arrhythmias predicated on interviews and making surface recordings in humans. This line of argument regarding safety in humans can be misleading; as we have shown in our experiments, immediately before and after the NID discharge there was no observable cardiac stimulation. However, if one is able to "see through" the electrical artifact during the discharge, cardiac stimulation was seen. Until intracardiac recordings can be made in humans with shielding to obliterate the electrical artifact that obscures possible intracardiac events, making safety conclusions in humans is premature. We did not state that NIDs cause ventricular fibrillation in humans, and we agree that we cannot conclude from our study that NID discharges cause arrhythmias in typical use.

We hope that readers agree that our study does suggest the possibility that NIDs may, in some circumstances, cause cardiac capture, and that this possibility should at least be considered in future research in humans. We hope that our work stimulates such research, using similar methods, in this area in humans.

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Reply

Although it is always best to test a device or a drug in humans, such testing is not necessarily feasible in all situations. Extensive tests, such as those performed to assess the effects of electrode positions and drug effects, are all but impossible except in an anesthetized animal model. Animal models have been used in many studies and have contributed to our understanding of many physiologic processes directly applicable to human beings. The pig model, for example, has been extensively used in studying defibrillation in internal or external defibrillators. Those findings have extrapolated well to human beings.

However, human studies have limitations. Thus, we (1) would differ from Dr. Pippin's broad indictment of animal studies as not being useful. Controversy exists in field reports of whether electrical stun guns have the potential, although infrequent, of directly causing ventricular arrhythmias. Human studies are limited in nature. Multiple exposures to the stun gun, prolonged exposure, seeking the most vulnerable thoracic sites, or safety margin of stun gun exposure would be impossible to perform in a human population. Monitoring of heart beats with transvenous electrodes would also be difficult to assess in a healthy group of human volunteers. The relationship of ventricular capture to induction of ventricular fibrillation (VF), for example, would have been difficult to assess in humans. The margin of safety and its relationship to thoracic position of the darts would also be difficult to assess. Although animals clearly do have a different anatomy, factors related to anatomic differences such as distance from the chest wall to the heart can be assessed.

We would also differ with Dr. Pippin's assessment that the cocaine findings are in contradiction to the common impression that cocaine causes arrhythmias. Cocaine may cause coronary spasm and an increase in sympathetic tone. Both of these effects can lead to arrhythmias. Our findings are clearly stated (1). Cocaine reduces the ability of a stun gun to induce VF. We were careful to note that the sedated state may minimize some of the sympathetic responses associated with cocaine ingestion. However, given the manner by which VF is induced with the stun gun impulses, via ventricular capture, it is quite physiologic to expect that cocaine would actually increase VF induction thresholds. This finding would have been difficult to confirm in a human study.

Finally, although one always has to be careful in extending results of animal studies to humans, such findings are extremely helpful in understanding mechanisms and in directing potentially acceptable studies in humans that would further our understanding and facilitate increased safety or efficacy of the tested object.

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